Amendments to the Specification:

Please amend the paragraph on page 1 following "CROSS-REFERENCE TO RELATED APPLICATIONS" as follows:

This application is a continuation-in-part of U.S. application ~~serial no. 09/740,676, filed December 18, 2000, pending, which is a continuation of~~ Serial No. 08/482,161, now U.S. Patent No. 6,162,461, which is a continuation-in-part of 08/454,121, now U.S. Patent No. 6,071,520, which is a §371 filing of PCT/NL94/00168, which is a continuation-in-part of 08/030,335, now U.S. Patent No. 5,491,073, which is a §371 filing of PCT/NL91/00165.

Please delete the paragraphs starting on page 30, line 1 through page 31, line 16.

Please replace the heading "EXPERIMENTAL PART" on page 8, line 14, with the following paragraphs and headings:

--BRIEF DESCRIPTION OF THE DRAWINGS

FIG. 1 shows the diagrammatic representation of the essential parts of the adenovirus adaptor vectors pMAd5 and pMab.

FIG. 2 shows the diagrammatic representation of the essential parts of the recombinant adenovirus adaptor vectors pMab-VP3 and pMab-con.

FIG. 3 shows the apoptin-induced apoptosis activity in 911 cells transfected with pMAb-VP3 or pCMV-VP3. Two independently cloned and purifies pMab-VP3 DNA-batches (pMab-VP3/ml and pMab-VP3-m^2) were used for the transfection of 911 cells. The cells were fixed 3 days after transfection and analyzed by indirect immunofluorescence using the apoptin-specific monoclonal antibody CVI-CAV-85.1 (85.1; Noteborn *et al.*, 1981). The percentage of cells that stained abnormally with DAPI is given as a relative measure for apoptosis.

FIG. 4 shows the apoptin (called VP3) –induced activity of human tumorigenic hepatoma HepG2 cells, osteosarcoma U2OS cells and normal non-transformed diploid FSK-1 keratinocytes infected with the recombinant-apoptin replication-defective adenovirus Ad-VP3. The cells were analyzed by indirect immunofluorescence using the monoclonal antibody 85.1 and stained with DAPI. The HepG2 and U2OS cells were fixed 1 day after transfection and the FSK-1 cells were harvested and fixed 4 days after transfection. The percentage of apoptin-positive

cells that stained abnormally with DAPI is given as a measure for apoptin-induced apoptosis (black boxes). As a control, the percentage of non-infected cells that have become DAPI-abnormally stained is given (open boxes).

FIG. 5 shows the apoptin- and/or VP2-induced apoptosis activity in Saos-2 cells transfected with 2.5 μg of pCMV-fs DNA expressing apoptin (formerly called pCMV-VP3; apoptin is named VP3) and 2.5 μg of pCMV-neoBam DNA (Danen-Van Oorschot, 1997); or with 2.5 μg pCMV-VP2 DNA expressing he CAV protein 2 (VP2), and 2.5 μg of 2.5 μg of pCMV-neoBam DNA; or with 2.5 μg pCMV-fs and 2.5 μg pCMV-VP2 resulting in the expression of both apoptin (VP3) and VP2. The cells were fixed 3, 4 and 5 days after transfection and analyzed by indirect immunofluorescence using the apoptin-specific monoclonal antibody CVI-CAV-85.1 or with monoclonal antibody CVI-CAV-111.1 (Noteborn and Koch, 1996). The percentage of cells that stained abnormally with DAPI is given as a relative measure for apoptosis.

FIG. 6 shows the diagrammatic representation of the essential parts of the recombinant adenovirus adaptor vectors pMab-VP2.

FIG. 7 shows the diagrammatic representation of the essential parts of the recombinant retrovirus transfer vector pLS-VP3-N.

DETAILED DESCRIPTION OF THE INVENTION